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# Forest Fires, Air Pollution and Mortality in Southeast Asia

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#### **Abstract**

In this paper, we assess the population health effects in Malaysia of air pollution generated by a widespread series of fires that occurred mainly in Indonesia between April and November of 1997. We describe how the forest fires occurred and why the associated air pollution was so widespread and long lasting. The main objective is to determine whether there were mortality effects and to assess how large and important these were. We also investigate whether the mortality effects were persistent or whether they simply represented a short-term, mortality harvesting effect. Our results show that the smoke haze from these fires had a deleterious effect on population health in Malaysia.

#### I. Introduction

Between April and November of 1997, a widespread series of forest fires in Indonesia threw a blanket of thick, smoky haze over a large portion of Southeast Asia. The smoke covered Indonesia, Malaysia, Singapore, and Brunei, as well as southern Thailand and the Philippines, and persisted for several months. The fires and smoke represented a major environmental disaster. The fires destroyed a large amount of rainforest, contributed to a significant release of greenhouse gases, and resulted in the loss of habitat for threatened or endangered species of plants and animals. They also led to adverse economic impacts through the destruction of commercial timber, plantations, and farmland, a reduction in tourism, the temporary shutdown of commerce, industry, and travel, and an increase in health care costs.

In this paper, we describe how the forest fires occurred and why the effects were so widespread and long lasting. The main focus is on assessing the population health effects of the air pollution generated by the fires. The objective is to determine whether there were mortality effects and to assess how large and important these were. We also investigate whether the mortality effects were persistent or whether they simply represented a short-term, mortality harvesting effect.

We examine the mortality effects in Malaysia due to smoke from the forest fires that occurred mainly in Indonesia. Data availability and the nature of the event introduce this interesting international dimension. The search for effects of the smoke haze in Malaysian cities several hundred miles from the fires suggests a minimum bound to the effects in Indonesia itself. The lack of appropriate health or mortality data for Indonesia means that air pollution effects cannot be evaluated satisfactorily in the areas where they are likely to have caused the greatest harm.

The 1997 Southeast Asian forest fires were unprecedented in terms of the area of land burned, the amount of smoke generated, and the size of the population living in areas affected by the smoke. However, they were not a unique event. Similarly severe fires occurred there in previous years—and also in subsequent years—as well as in other places around the globe that same year, including Brazil and Mexico. What distinguished the 1997 occurrence of forest fires in Southeast Asia were their intensity and duration as well as the large population that was affected by the smoke.

There are a host of factors that explain the occurrence of forest fires in Indonesia in 1997. As with the occurrence of large-scale environmental degradation in other settings, some direct or contributing causes are demographic. These include population pressure associated with rapid rates of population growth and specific patterns of migration and resettlement. These factors—and others—have contributed to rapid rates of deforestation, but local land clearing practices and policies regarding the management of natural resources appear to have played a central role. The relative importance of the various contributing causes, and the specific role of demographic factors, is not well understood in Indonesia or in other settings (Palloni, 1994).

There are several important policy issues associated with this study. One concerns the reforms and policy changes in Indonesia that are necessary to reduce the likelihood of a recurrence of this event. These need to be based on an understanding of the social, economic, environmental, and policy reasons behind this disaster. A second policy issue concerns the appropriate responses to this type of event, at the local, regional, and global levels. At the local and regional levels, the forest fires and smoke haze were associated with adverse economic

impacts and possible deleterious health effects. At the regional level, the transboundary incidence of health, economic, and environmental effects raise important issues regarding politics, international relations, and security and conflict. Relatively little is known about the effects of environmental disasters in one nation on economic and health outcomes in neighboring states. Part of the reason is that the cross-country effects of environmental degradation are generally slow-acting, and are therefore of uncertain magnitude and severity. Finally, at the global level, this event was associated with the destruction of a massive area of rainforest, huge emission of greenhouse gases, and future global warming.

We return to the policy issues raised by this study in the final section of the paper. We begin, in the next section, by describing the forest fires in Southeast Asia in 1997. This is followed, in Section III, by a brief review of the relationship between air pollution and health outcomes from previous studies. In Section IV we describe the data on air pollution and mortality and in Section V we discuss the main modeling issues.

Our results, presented in Section VI, provide clear evidence of mortality effects of the smoke haze caused by the forest fires. We find that the effects of a high air pollution day associated with the smoke haze are to increase total all-cause mortality by 22 percent. Increased mortality is apparent in two locations—Kuala Lumpur and Kuching—and affects mostly the elderly. In Kuala Lumpur, non-traumatic mortality among the population aged 65-74 increased 72% following a day of high air pollution levels. This effect was persistent—it was not simply a moving forward of deaths by a couple of days. This suggests that there were real and serious health effects of the smoke haze.

#### II. Forest Fires in Southeast Asia

Fires to clear forest and brush have been an annual occurrence in Indonesia for generations. When land clearing was limited, such as with shifting agriculture, the effects were small. In the past 15 years, however, the scale of land clearing has increased, as large-scale rubber and oil palm plantations emerged and used burning as an inexpensive method of clearing vast areas (Brauer and Hisham-Hashim, 1998). The earliest reports of regional biomass smoke date back to 1982, with additional episodes reported in 1986, 1991, and 1994 (Brauer and Hisham-Hashim, 1998). The fires that occurred in 1997 were by far the worst ever. The forest fires and smoke were concentrated during the intermonsoon dry season of July to October, when land is cleared before planting and during which weak Southeast winds predominate (Nichol, 1998). Once the forest fires were started, they could not be put out easily. Rather, it was only the arrival of the seasonal rains that finally extinguished them.

The fires were concentrated in the Indonesian provinces of Sumatra and Kalimantan. However, fires also occurred in Irian Jaya, Sulawesi, Java, Sumbawa, Komodo, Flores, Sumba, Timor, and Wetar, and in Malaysian and Bruneian parts of Borneo. In total, the 1997 fires burned 2-3% of Indonesian land area (Levine et al., 1999; Liew et al., 1998). Using SPOT satellite images, Liew et al. (1998) estimated that the total burned area in Kalimantan and Sumatra was 45,600 square kilometers (km²). In these two provinces, agricultural and plantation

<sup>&</sup>lt;sup>1</sup> Indonesia has a land area of approximately 2 million square kilometers. Approximately 60% of Indonesia's land is forested; it contains over 10% of the world's rainforest and about 40% of rainforest in Asia. Indonesia ranks third in rain forest area, after Brazil and Congo.

<sup>&</sup>lt;sup>2</sup> Liew et al. (1998) estimated the burned area to be 30,600 km<sup>2</sup> in Kalimantan (total area 539,460 km<sup>2</sup>) and 15,000 km<sup>2</sup> in Sumatra (total area 473,606 km<sup>2</sup>).

areas accounted for 50% of actual burned area; forest and bushes, 30%, and peat swamp forest, 20% (Liew et al., 1998).

#### Causal Factors

The forest fires in Southeast Asia in 1997 were entirely man-made. The main reason for setting the fires was to clear the land of vegetation—either primary growth forest or overgrowth—for shifting agriculture, plantations, or for transmigration project settlers. Fires were often used to settle land disputes and, in particular, to drive off settlers. Although originally blamed on slash-and-burn farmers, examination of satellite images have shown that large plantation companies, many with ties to the Suharto government, used the fires to clear vast areas of land (Business Times, 1997; Economist, 1997a). The land has been used to plant trees for rubber, timber (for pulp and paper), and palm oil. Plantations generally burned marginal land that had already been logged, while slash-and-burn farmers and the transmigration project settlers often burned primary forest. Burning provides a cheap and fast method for clearing land and is efficient for those doing the land clearing.

Although there have been no detailed studies of structural causes of the fires in Southeast Asia in 1997 or other years, there are various causal factors that are likely to be important. Among the principal influences on Indonesian deforestation are demographic factors; government policy and its enforcement; and political, social, and legal institutions. These forces generally act in combination with each other. Of particular concern is the role of population processes, although causal factors tend to operate in combination with each other. Growing population size and density as well as migration and settlement patterns are potentially important determinants of deforestation. However, Palloni's (1994) literature review suggests that other factor are likely to be more critical than population; in particular, the effects of population have not been demonstrated convincingly in the existing literature. Nevertheless, slower population growth would reduce pressure on the environment and might provide the room to develop policies and institutions to protect the environment (National Research Council, 1986).

National economic policy in Indonesia—epitomized by the Transmigration Program—favors the extensive utilization of unused resources and, especially, underpopulated land. The Transmigration Program emerged as a response to rapid rates of population growth and uneven population distribution in Indonesia (Fearnside, 1997). The goal of the program has been to move approximately half a million people a year from the densely settled islands—Java and Bali, in particular—to sparsely settled areas in Sumatra, Kalimantan, and Irian Jaya. This program requires an infrastructure of roads and transportation to open up new areas and a large infusion of capital for enterprises and settlement. Alternative development models that promote agricultural intensification, rather than extensification, may be more sustainable. The Transmigration Program has selected destination sites that are of questionable agricultural value. For instance, the remote peatlands of Riau and Jambi provinces in Sumatra and of southern Kalimantan are major transmigrant destinations. However, the peatland environment is of questionable agricultural value—especially since migrants do not know how to farm it (Nichols, 1989).

The management of natural resources is another important causal factor that reflects the role of government policy. In Indonesia, the central government owns and manages all of the country's forests. Management occurs through the design and enforcement of specific policies and regulations, such as logging concessions, property rights, land tenure, and the use of fires to clear land. Political factors are potentially important here, as demonstrated by concessionaires'

ties to the former president, Suharto. Although the use of fire was been banned in 1995—as a result of the 1994 fires—this law has not been widely enforced and hence has had little effect. Technology may play a role in the effective use of natural resources; especially relevant are technological alternatives to fire for converting marginal land to agricultural or plantation use. For instance, there are more efficient methods available for clearing the land of overgrowth in preparation for tree planting for plantations that require greater up-front investment and a longer time horizon. However, the design of the logging and plantation concessions often does not support their use.

Other potentially important factors include political, social, and legal institutions. Forest fires represent a failure of these institutions. An institution that is likely to be of particular importance is land tenure. Most fires occurred in areas with joint land ownership (Nichol, 1998). The lack of clear land tenure laws, uncertain land status, and poor relationships between concessionaires, migrants, and local people all contributed to the fires being started intentionally (Levine et al., 1999). In Indonesia, although the forests are government owned, local people have easy access and use forests as their own resource. Traditional *adat* law, which has governed the use of forest lands until the past few decades, clashes with more recent logging concessions handed out by the government in Jakarta. The arrival of concessionaires has been associated with restrictions for locals on use of the forests, with little account paid to traditional, albeit informal, land rights. Burning is used as a weapon by both sets of claimants. Small farmers sometimes burn trees planted by big forestry companies, and large firms have in turn burnt land to drive out small landholders. The rise of tree plantations is seen by some as the most powerful force behind the conversion of forest lands in Sumatra and Kalimantan (Schweithelm and Glover, 1999).

In addition to the causal factors discussed above, there were three important contributing factors that affected the severity and duration of the fires in 1997. First was the presence of the El Niño Southern Oscillation (ENSO) phenomenon, associated with warming of the waters of the equatorial Pacific Ocean. ENSO led to unusual weather patterns around the world and, in particular, to a severe drought in Southeast Asia. ENSO delayed the northeast monsoonal weather pattern, which let the fires burn several months longer than usual (Brauer and Hisham-Hashim, 1998). The drought in 1997 was said to have been the worst in 50 years (New York Times, 1997). The effect of ENSO, however, was to exacerbate the fires that were being set, not to cause the fires themselves.

A second contributing factor was the peat deposits that cover the topsoil in parts of Sumatra and Kalimantan. The peat deposits can be as deep as 2 to 3 meters. Peat is a mixture of decaying organic matter, such as roots, tree branches, and leaves. Generally, it is a poor soil for farming, although areas with peat soils are important destinations for the Transmigration Program. During periods of prolonged drought, the peat dries and can combust readily. It burns easily and spreads fire quickly, usually underground. Smoldering peat can burn at depths of up to 2 meters, which makes it difficult to extinguish. Only heavy rainfall can put out the smoldering fires. Burning peat also emits larger amounts of smoke than the burning of other forms of biomass. Finally, seams of coal near the surface have also caught fire and may smolder for a long time. Some coal seams that caught fire during the early 1980s are still smoldering.

#### Smoke Haze from Forest Fires

The smoke from the forest fires traveled across the Southeast Asian region, reaching all the way to southern parts of Thailand and the Philippines but with the most severe effects being felt in Singapore, Malaysia, and Brunei. Of course, the smoke in Indonesia itself was tremendous, although Java, with 115 million people, was mostly spared due to prevailing winds. Nevertheless, the population of the affected area was about 70 million people.

The ENSO phenomenon that led to drought conditions in Southeast Asia was also associated with high pressure over the region. This prevented dissipation of the smoke and caused it to spread into relatively thin layers of great horizontal extent while maintaining its concentration (Andreae, 1990). When the smoke reached urban areas, it often led to an atmospheric inversion, trapping emissions from cars and factories and thereby multiplying the negative health effects.

The smoke haze from the forest fires lasted from late July to December 1997. Little could be done to combat the smoke haze once fires were raging. The air pollution subsided only after the arrival of the monsoons. During the peak period of haze in September 1997, the ambient air pollution concentrations in Kuching, in Sarawak, Malaysia, reached 930 micrograms per cubic meter ( $\mu$ g/m³), an astonishingly high level more than 10-times higher than normal (WHO, 1998; Brauer and Hisham-Hashim, 1998). In many other cities in the region, air pollution indexes repeatedly reached unsafe level. During periods of severe air pollution, schools, factories, and offices were closed and people—especially children, the elderly, the sick, and the infirm—were advised to stay indoors and restrict their activities. Facemasks were also distributed, though their use probably had little benefit. People who could leave the area, including many foreign nationals, did so. Nevertheless, in Indonesia at least, reports indicated that many people viewed the smoke haze as a nuisance, rather than as a major environmental disaster and possible health hazard (Kristof, 1997).

Interestingly, government reactions were muted. For example, Singapore's *Straits Times*, whose editorial views usually closely reflect the government's, criticized one Indonesian minister for calling the fires a natural disaster, pointing out that they were clearly an avoidable man-made disaster. In Malaysia, an opposition party urged the government to demand compensation from Indonesia (Economist, 1997b), although it did not do so. The rather staid reaction of governments in the affected countries clearly reflects the tone of regional diplomatic relations. Nevertheless, the tone could change in the future if the fires and smoke haze become an annual occurrence.

The atmospheric pollutant that most consistently increases with biomass smoke is suspended micro-particulate matter. These small solid combustion particles comprise of organic matter, black elemental carbon (soot particles), and inorganic materials such as potassium carbonate and silica (Andreae, 1990). Levine (1999) identified the main gases produced during biomass burning process as carbon dioxide (CO<sub>2</sub>), carbon monoxide (CO), methane (CH<sub>4</sub>), oxides of nitrogen (NO and NO<sub>2</sub>), and ammonia (NH<sub>3</sub>). Methane, carbon monoxide, and oxides of nitrogen lead to the photochemical production of ozone (O<sub>3</sub>) in the troposphere. Carbon dioxide, methane, and ozone are greenhouse gases that can lead to global warming. In addition, particulates absorb and scatter incoming solar radiation and, hence, can impact the climate on a local, regional, and global scale. Finally, biomass burning further contributes to increases in atmospheric carbon dioxide (and other greenhouse gases) by destroying forests that would

otherwise help to naturally eliminate these gases.<sup>3</sup> Global warming itself may be implicated as a cause of the ENSO phenomenon, representing a vicious circle.

Andreae (1990) estimated that global biomass burning contributed as much as 40% of gross carbon dioxide emissions and 38% of tropospheric ozone. One analysis of the emissions associated with the Indonesian forest fires indicated that in just the two main areas (Kalimantan and Sumatra) about 200 Mt of carbon were released. This represents 10% of the global carbon dioxide emissions associated with biomass burning and 3% of global carbon dioxide emissions (Levine, 1999; Andreae, 1990).<sup>4</sup>

An analysis of smoke particulates in Kuala Lumpur during October 1997 indicated that they originated from fires in Southern Kalimantan (von Hoyningen-Huene et al., 1999), though at other times the predominant source was the fires in Sumatra. A separate study of air quality in Kuala Lumpur found that the smoke haze was associated with high levels of suspended microparticulate matter, but with relatively low levels of other gaseous pollutants such as carbon monoxide, nitrogen dioxide, sulfur dioxide, and ozone (Noor, 1998; Awang et al., 2000).

#### III. Health Effects of Air Pollution

There are possible short-term and long-term health effects of exposure to air pollution. In the short term, high levels of air pollution lead to acute conditions, such as respiratory infections, and mortality, including deaths from accidents as well as from chronic conditions. In addition, blockage of sunlight may promote the spread of harmful bacteria and viruses that would otherwise be killed by ultra-violet B (Beardsley, 1997). The possible long-term health effects of exposure to air pollution are unknown and difficult to detect. Components of smoke haze, including polycyclic aromatic hydrocarbons, are known carcinogens whose effects may not be apparent for years. The consequences may be more severe for children, for whom the particulates inhaled are high relative to body size and who may be passing through critical periods of development.

There have been two types of studies that have linked daily concentrations of ambient particulate matter to mortality. One has examined the health effects of high concentrations of air pollution—such as the killer fogs that struck London in 1952, the Meuse Valley in Belgium in 1930, and Donora, Pennsylvania in 1948 (see Pope, Dockery, and Schwartz, 1995). The fog in London in December 1952 raised deaths over the course of a week to almost three times their usual value, from 945 deaths during the week of 6 December to 2,484 deaths during the week of 13 December (Schwartz, 1994b). Many of the deaths were due to chronic obstructive pulmonary disease and cardiovascular disease and were concentrated among the elderly. In our analysis, we search for mortality effects associated with a large increase in air pollution in various cities in Malaysia. However, we expect these to be of much smaller magnitude than the "killer fog" type of effect.

<sup>&</sup>lt;sup>3</sup> The forest fires also contributed to a reduction in biodiversity through species extinction and habitat loss. For instance, Curran et al. (2000) showed that the effects of ENSO and logging in Kalimantan together led to a drastic reduction in the regenerative capability of an important rainforest tree species.

<sup>&</sup>lt;sup>4</sup> Global emissions of carbon dioxide correspond to the release of 7,000 million tons (Mt) per year of carbon (Andreae, 1991). Of this, 1,800 Mt per year—or about one-quarter—was associated with tropical deforestation (Andreae, 1991). Biomass burning associated with tropical deforestation also contributes significantly to the release of two other greenhouse gases, ammonia (CH<sub>4</sub>) and nitrogen dioxide (NO<sub>2</sub>). Annually, 11% of ammonia emissions and 13% for nitrogen dioxide emissions are associated with biomass burning (Watson et al, 1990; National Academy of Sciences, 1991).

The second type of study to examine air pollution and mortality has considered the effects of moderate levels of air pollution, such as those that are currently experienced in urban areas of the U.S. and elsewhere. These studies have found a clear relationship between air pollution and daily mortality. Especially strong effects are found for suspended microparticulate matter, the principal component of smoke haze; there is little apparent confounding by sulfur dioxide or other gaseous pollutants. Previous studies have reported no threshold effects, although the evidence supporting the conclusion that the health effects are similar at high and low pollution levels is not clear-cut. Findings from studies in a variety of different locations have found the dose-response relationship to be relatively stable across study sites. However, there have been relatively few studies outside of North America and Europe. A recent meta-analysis reported an estimated risk ratio of 1.06 for a 100  $\mu$ g/m³ increase in total suspended particulates (Schwartz, 1994a).

There are several unresolved issues concerning whether the relationship between  $PM_{10}$  and mortality is causal (Samet et al., 2000). Two that are particularly relevant to this study are understanding *individual* exposure to air pollutants and estimating the burden of premature death.<sup>6</sup> Regarding the first issue, air pollution concentration data from area monitors are often used as a proxy for individual exposure. An important question concerns the extent to which this leads to valid inferences. For this study, the relationship between air pollution and mortality may be affected by behavioral responses to episodes of smoke haze. These may include more time spent indoors rather than outdoors, use of masks and air conditioning, and reduced activity levels, all of which were recommended by the government following alerts of high air pollution levels. As in the more general case, measurement error associated with differences between personal exposure and ambient pollution levels leads to attenuation in the size of the estimated effects (Zeger et al., 2000). However, studies drawing on individual-level data on exposure suggest that the magnitude of this bias is likely to be small (Dominici, Zeger, and Samet, 2000).

The burden of premature death associated with air pollution depends on the magnitude of harvesting effects. The issue can be understood best by considering a simple model used by Samet et al. (2000) and others. Suppose there are two groups in the population, the frail and the non-frail. Normally, deaths occur only among the frail group, which is replenished each period by people transitioning from the non-frail group. A pollution event that increases the number of deaths may deplete the pool of frail persons and lead to fewer deaths on subsequent days. In this situation, the mortality burden is small, totaling relatively few person-days—and even fewer person-days of *healthy* life. Alternatively, a pollution event may lead to some deaths among the non-frail, in which case the burden of premature death would be considerably larger.

Harvesting is a key issue and a major point of controversy in understanding the health effects of air pollution. The two prospective cohort studies of particulate air pollution and daily deaths conducted to date, the Harvard Six Cities Study (Dockery et al., 1993) and the Cancer Prevention Study (Pope et al., 1995), have found significant long-term displacement of deaths, perhaps on the order of one to two years. Results emerging from recent studies that apply new analytical techniques to standard time series mortality and air pollution data (see Zeger,

<sup>&</sup>lt;sup>5</sup> Bascom et al. (1996a, 1996b) and Dockery and Pope (1994) review this literature.

<sup>&</sup>lt;sup>6</sup> The other issues concern uncovering and understanding differences in results across study sites; whether there has been adequate consideration of potential confounders such as other pollutants; and whether different analytical methods yield similar results (Samet et al., 2000).

Dominici, and Samet, 1999; Schwartz, 2000) indicate that this relationship cannot be attributed solely to harvesting effects. Despite these results, this issue is far from resolved.

A final matter concerns the effects of air pollution exposure on health status, rather than mortality. A significant mortality effect is likely to be indicative of acute morbidity effects, regardless of whether the mortality effect is of a short or long duration. The presence of mortality effects thus serves as a sentinel indicator for other correlated—but unmeasured—morbid outcomes.

Recent studies in the region have examined possible health effects of the 1997 forest fires. Some findings of negative health effects have emerged in Malaysia (WHO, 1998). For example, outpatient visits in Kuching, Sarawak, increased between two and three times during the peak period of smoke haze, and respiratory disease outpatient visits to Kuala Lumpur General Hospital increased from 250 to 800 per day. Effects were found to be greatest for children, the elderly, and people with pre-existing respiratory problems. Suggestive data were assembled that indicated an increase in cases of asthma, acute respiratory infections, and conjunctivitis during August-September 1997 at a number of major hospitals in Kuala Lumpur (Brauer and Hisham-Hashim, 1998). Similar findings were reported for the state of Sarawak and for Singapore. In Singapore, an increase in levels of micro particulate matter (PM<sub>10</sub>) from 50 to 150 µg/m<sup>3</sup> during the last week of September 1997 was associated with a 12% increase in cases of upper respiratory tract illness, a 19% increase in cases of asthma, and a 26% increase in cases of rhinitis, based on health surveillance data from clinics (WHO, 1998). There were, however, no significant increases in hospital admissions or mortality. Studies such as these of healthseeking behavior or diagnoses based on visits to health facilities suffer from unknown selection effects. For example, the publicity associated with the smoke haze may have led to greater health care seeking behavior. It may have led to more diagnoses through the heightened awareness of symptoms among both patients and health care providers. Finally, it is important to note that there are no data or studies available for Indonesia, the country most affected by the forest fires and smoke.

#### IV. Data

Data for this study were assembled from several different sources. Mortality data come from Malaysian vital statistics records. Information on air quality for the capital city, Kuala Lumpur, was obtained from daily measurements made by the Malaysian Meteorological Bureau. Climate data was extracted from the U.S. National Oceanic and Atmospheric Administration's Global Weather Station Database.

The mortality data comprise of individual records, with information on age, sex, and cause of death, for the period 1994-97. Cause of death is coded according to the Ninth revision of the International Classification of Disease (ICD-9) and a single, primary cause of death is recorded. Information was obtained on deaths in several major Malaysian cities, including Kuala Lumpur, Kuching, Johore Bahru, Penang, and Ipoh. Malaysian vital statistics data are recognized to be of generally high quality. However, as explained below, there are some possible shortcomings in the quality of the cause-of-death classification for Kuala Lumpur.

In Table 1 we present for Metro Kuala Lumpur summary statistics covering several different mortality measures. Metro Kuala Lumpur includes the federal territory that is the capital of Malaysia, but none of the surrounding areas. The population of Metro Kuala Lumpur in 1994 was estimated to be 1.7 million, while Greater Kuala Lumpur included 2.5 million

people. The mean number of daily deaths in Kuala Lumpur is 16.1. This relatively small number is a consequence of the low crude death rate of Malaysia. At 4.5/1000, the crude death rate for Malaysia is about half the value in the U.S. and reflects the fact that Malaysia has a relatively young population and low mortality rates at each age. In particular, the infant mortality rate in Malaysia is 8.3 per 1,000 live births and life expectancy is 70 for men and 75 for women. The row showing non-traumatic deaths removes those due to external causes such as accidents and injuries (ICD-9 codes E800-E999). These are further divided into three categories: deaths due to cardiovascular disease, respiratory disease, and other causes. Exposure to air pollution is expected to increase the number of daily deaths due to cardiovascular and respiratory disease. The final panel in Table 1 shows the number of deaths by four age categories: less than one year, 1-64 years, 65 to 74 years, and 75 and older. Air pollution effects are expected to be concentrated among infants and the elderly.

Deaths due to undetermined causes comprise a larger proportion in Kuala Lumpur than in other Malaysian cities. In Kuala Lumpur, 17% of deaths were classified under the ICD grouping "Symptoms, Signs, and Other Ill Defined Conditions." By comparison, this category contained only 3% percent of deaths in Kuching. The proportion of deaths with ill-defined causes rises with the decedent's age in Kuala Lumpur: among the very old (age 75+), almost half the deaths—47%—were in this residual category. In contrast, this residual category accounted for 4% of deaths to those under 65 years of age and 15% to those aged 65-74. According to the Malaysian government statisticians, this is due to unascertained causes, respiratory failure with no mention of any specific cause, senility, and pyrexia of unknown origin. A consequence is that we are unable to undertake a detailed analysis of the relationship between air quality and cause of death for Kuala Lumpur.

The Malaysian Meteorological Bureau provided data on average daily concentrations of suspended micro-particulate matter for Kuala Lumpur for the period 1996-97. The specific measure available is the concentration of micro-particulates of diameter less than 10 microns  $(PM_{10})$ .

Climate data were extracted from the Global Weather Station Database, which is assembled by the National Climatic Data Center of the U.S. National Oceanic and Atmospheric Administration. This database draws on weather data that are collected routinely at weather stations around the world. The stations in Malaysia and elsewhere are principally at airports. The database includes measures of temperature, humidity, precipitation, wind speed and direction, and visibility. These data covered the period 1994-97. Visibility provides an alternative—though somewhat less sensitive—measure of air quality.

In Table 2 we present summary statistics for weather and air quality in Kuala Lumpur for the period 1996-97. Malaysia is located near the equator and there is thus little annual variation in temperature or dew point (a measure of humidity). The average daily  $PM_{10}$  level for this period is 64.2  $\mu g/m^3$ , which is higher than the recommended level of 50  $\mu g/m^3$  established as the U.S. National Ambient Air Quality Standards for  $PM_{10}$  by the U.S. Environmental Protection Agency (EPA) in 1987. Also note that at the upper end of the distribution, there are a number of

<sup>8</sup> 10 microns is 1/100 of a millimeter, which is roughly 1/7<sup>th</sup> the width of a human hair.

<sup>&</sup>lt;sup>7</sup> Cardiovascular disease covers ICD-9 codes 390-448 (heart disease, hypertensive diseases, ischemic heart disease, diseases of pulmonary circulation, cerebrovascular diseases, and arterial disease); the respiratory disease category covers ICD-9 codes 480-487 (pneumonia and influenza), 490-496 (chronic obstructive pulmonary disease and allied conditions), and 507 (other respiratory causes).

days for which the average  $PM_{10}$  level in Kuala Lumpur exceeds the maximum EPA recommended concentration for a 24-hour period of 150  $\mu g/m^3$ . For comparison, the mean  $PM_{10}$  concentration for Los Angeles was 33  $\mu g/m^3$  in 1997 and there were no days on which it exceeded 150  $\mu g/m^3$  (Environmental Protection Agency, 1998).

Figure 1 shows  $PM_{10}$  levels for Kuala Lumpur for the two-year period of 1996-97. Dramatically visible in this figure is the spike in  $PM_{10}$  levels at the end of 1997, brought about as the result of the forest fires in Indonesia. A second measure of air quality in Table 2 is the mean daily visibility in kilometers. Figure 2 shows a graph of the reciprocal of visibility over the entire four-year period for which daily mortality data are available. Two spells of poor visibility are apparent in Figure 2. The first corresponds to the forest fires that occurred in the Fall of 1994 while the second matches the fires in 1997 that are captured in the  $PM_{10}$  measure.

#### V. Modeling Issues

The modeling approach follows closely the current standard in the literature for studying the effects of air pollution on daily mortality (see Samet, Zeger, and Berhane, 1995). The goal is to estimate the percent change in daily mortality from a change in the (lagged) air pollution measure. Poisson regression is appropriate for modeling  $y_t$ , the count of deaths occurring on day t, because the daily counts are a set of non-negative integers. One relevant property of the Poisson distribution is that the mean is equal to the variance. This reflects the fact that the variability of the count of daily deaths tends to increase with the mean number of daily deaths.

We model the logarithm of the mean daily mortality count—and the variance of daily deaths—as a linear function of a vector of covariates,  $x_i$ :

$$\log(\mu_t) = \log(E(y_t)) = x_t' \beta.$$

Each element of  $\beta$ , the vector of estimated regression coefficients, represents the log of the mortality rates for two days that differ only with respect to a one-unit change in the corresponding covariate; exponentiated parameter estimates are interpreted as relative risks.

This model is straightforward to estimate and interpret. There are, however, two potential methodological issues that may affect the results. First, the variance of daily mortality may exceed the mean, a situation known as overdispersion. This may arise due to the effects of unmeasured factors that are excluded from the model, such as the occurrence of an infectious disease epidemic. Second, mortality counts on neighboring days may be more closely related to each other, either directly or inversely, than counts on days further apart in time. The presence of autocorrelation, as this phenomenon is known, may also reflect the effects of unmeasured factors.

To address the problem of overdispersion, we assume that the variance is proportional to the mean, rather than equal to the mean. We assume a time-invariant constant of proportionality,  $\phi$ , such that

$$var(y_t) = \phi \mu_t.$$

We estimate  $\phi$  and use it to adjust the standard errors for the estimated parameters.

To account for autocorrelation, we adopt the generalized estimating equation approach (GEE) of Liang and Zeger (1986). GEE provides a general method to account for the correlation

between observations in generalized linear models, such as the Poisson model. Note that GEE models are *marginal* models, in which the marginal expectations (that is, the average response for observations sharing the same covariates) are modeled as a function of explanatory variables.

Under the GEE approach, we estimate  $\hat{\beta}$  by solving the generalized estimating equation:

$$\frac{\partial \mu'}{\partial \beta} V^{-1}(y - \mu(\beta)) = 0.$$

The covariance matrix V is unknown, and we must model it in order to estimate the parameters  $\hat{\beta}$  efficiently and obtain corrected standard errors. We model V as

$$V_A = A^{1/2} R(\alpha) A^{1/2},$$

where A is a diagonal matrix with elements  $var(\hat{y}_t) = \hat{\mu}_t$  that are weighted by the estimate of overdispersion,  $\hat{\phi}$ . The working correlation matrix  $R(\alpha)$  captures the autoregressive process of order q (AR-q). The AR-q process can be written as

$$u_t = \alpha_1 u_{t-1} + \cdots + \alpha_q u_{t-q} + a_t,$$

where the terms  $a_t$  are independent with zero mean. The parameters  $\alpha_1,\ldots,\alpha_q$  are estimated using the Pearson residuals  $\hat{u}_t = (y_t - \hat{\mu}_t)/\sqrt{\mathrm{var}(\hat{\mu}_t)}$  and linear regression. The elements of  $R(\alpha)$  are calculated from these estimated parameters. With an AR-1 process the elements of  $R(\alpha)$  are equal to 1 along the diagonal and  $\alpha^{|t-s|}$  for the off-diagonals.

This model assumes that the correlation between two mortality counts,  $y_t$  and  $y_s$ , depends only on the time between the observations t and s. Note that this correlation may also depend on their means,  $\mu_t$  and  $\mu_s$ ; however, in this case the process would be non-stationary and difficult to estimate. We assume that the data followed a low-order autoregressive process and experimented with autoregressive processes of varying order to assess the sensitivity of our results.

Because V is unknown and is approximated by an estimate,  $V_A$ , there are a couple of possible sources of error. First, the correlational structure represented by the AR-q time series process may in fact be nonstationary, in which case the parameter estimates will be inefficient. Second, the model-based variance estimates for  $\hat{\beta}$  that we report are the GEE equivalent of those based on the inverse of the Fisher information matrix. These variance estimates are consistent when the model is correctly specified. The alternative is to use the empirical variance estimates. The empirical estimates are asymptotically unbiased, but may be highly biased when the number of clusters is small—as would be case here since we have only a single time series. On the other hand, the model-based variance estimates that we use have better properties when the number of clusters is small, even if the working correlation matrix,  $V_A$ , is misspecified.

Covariates that are included in all models are weather terms—temperature and a measure of humidity; seasonal terms; and controls for longer-term trends in mortality or population that

<sup>&</sup>lt;sup>9</sup> Also known as the sandwich, robust or Huber/White variance estimates.

are unlikely to be related to air pollution. Factors within the study area that are likely to remain constant—or change only slowly over time—are not included in the models and are picked up by the time trend. These include average health status, the prevalence of health related behaviors such as smoking, access to and use of medical care, time spend outdoors, occupation, income, and housing characteristics such as crowding or air conditioning. We omit detailed discussion of the results concerning these covariates in the next section and focus instead on the effects of high levels of air pollution associated with the smoke haze.

#### VI. Results

We begin the results section by examining the effects of the smoke haze on mortality for Kuala Lumpur. For this city we have two air pollution measures, PM<sub>10</sub> and visibility, to identify the periods of severe smoke haze. The PM<sub>10</sub> data, representing our best measure of air pollution, cover the period 1996-97 while the visibility data are available for the four-year period of 1994-97. We next look at the effects of smoke haze on daily mortality for other Malaysian cities, using visibility-based measures alone. We end by reexamining our main results using autoregressive models and investigating the cumulative lagged effects of exposure to the smoke haze over periods of several days and weeks.

Before turning to a detailed presentation of the results, we discuss several model specification issues. These concern the indicator of exposure to smoke haze and the age groupings that we use. The regression results focus on the one-day lagged effects of high smoke haze levels. Our choice to use a one-day lag was based on a preliminary analysis that found the clearest and most consistent effects emerging with this measure. It also matches much of the literature in this field. Substantively, the one-day lagged effects suggest that the smoke haze has a nearly immediate initial impact on health status. Later in the paper we examine how mortality is affected by longer lagged values of air pollution.

Our best measure of smoke haze levels is  $PM_{10}$  and an exploratory analysis indicated that strongest mortality effects of the smoke haze emerged when  $PM_{10}$  concentrations exceed 210  $\mu g/m^3$ . There are a total of 12 of these days over the two-year period 1996-97. Although this cut-off does not have obvious substantive significance, it clearly identified high air pollution days associated with the smoke haze from the Indonesian forest fires. We settled on a categorical indicator of exposure to high levels of smoke haze because we found evidence of non-linear effects (described below). The construction of a categorical indicator also allows us to calibrate the  $PM_{10}$ -based measure of air pollution with one based on visibility. In particular, the cut-off used to identify high air pollution days based on  $PM_{10}$  levels can be translated into a cut-off based on visibility levels. This cut-off can then be applied to the longer time-series of data for Kuala Lumpur as well as data for other Malaysian cities.

The presence of non-linear effects is an unresolved issue in much of the literature that looks at the short-term effects of air pollution (Samet, Zeger, and Berhane, 1995) but is quite clear here. To illustrate this, in Table 3 we compare the one-day lagged effects of a high smoke haze day ( $PM_{10} > 210~\mu g/m^3$ ) on mortality due to non-traumatic causes among residents of Kuala Lumpur aged 65-74 using a continuous and a discrete measure of air pollution. The results based on the discrete measure (shown in the right column) indicate a relative risk of 1.72 associated with a high air pollution day, which is roughly twice as large as the relative risk of 1.39 associated with the continuous measure (left column). This suggests that non-linear effects are important. However, estimating the shape of the non-linear effects is not the focus of our

analysis and would lead to unnecessarily complicated models. The categorical indicator offers us a simpler interpretation for the central results. The continuous and discrete effects are not significantly different in the statistical sense and are qualitatively similar. Note also that the size of the continuous  $PM_{10}$  effect for Kuala Lumpur is similar to that found elsewhere (see Schwartz, 1994).

Finally, our analysis examines mortality effects for four age groups: under 1 year of age, 1-64 years, 65-74 years, and age 75 and above. These age groups correspond to those used published studies and focus attention on deaths at the youngest and oldest ages, where the effects of exposure to air pollution are likely to be strongest. Choosing different age categories does not alter the results that we report below. More generally, with only one or two exceptions (mentioned below), our results show little sensitivity to the exact specifications that we choose.

#### PM<sub>10</sub> Models for Kuala Lumpur

In Table 4 we show the estimated mortality effects in Kuala Lumpur of a high air pollution day associated with the smoke haze from the Indonesian forest fires. A high pollution day is defined here as one with a  $PM_{10}$  level of  $210~\mu g/m^3$  or above. The parameters in the table are relative risks. Thus, the first entry in the table indicates that a high air pollution day is associated with a relative risk of 1.22 for mortality due to all causes and across all ages. This effect is significant at the five-percent significance level. The columns in Table 4 show effects for total mortality, non-traumatic mortality (which removes deaths due to accidents and injuries), and mortality due to cardiovascular disease, respiratory disease, and other causes. The rows show the effects across all ages and for age groups <1, 1-64, 65-74, and 75 plus.

The first pattern apparent from Table 4 is that there are no estimated parameters shown for deaths due to cardiovascular and respiratory causes. These results are omitted because the estimated models do not fit the data and there are no significant regression coefficients. This is due primarily to data quality shortcomings concerning the coding of cause of death in Kuala Lumpur discussed above.

Table 4 shows that for the entire population, deaths due to non-traumatic causes are 21% higher after a high pollution day and this effect is statistically significant at the ten-percent level. Mortality due to non-traumatic causes is the best overall measure to focus on because the effects of accidents and injuries (which together comprise traumatic mortality) could reasonably be positively or negatively associated with the smoke haze. Substantively, the most important pattern in the table is the concentrated mortality effect on people aged 65-74. On the day after a high air pollution episode, all-cause mortality increases by 57% and deaths due to non-traumatic causes increase by 72%. Deaths due to non-traumatic causes other than cardiovascular and respiratory factors are also significantly higher, but these effects are somewhat difficult to interpret given the problems with cause of death coding. Finally, high air pollution days are consistently associated with increased mortality for those aged 75 and over, but these effects are relatively modest and are statistically insignificant.

#### Visibility Models for Kuala Lumpur

Using information on visibility to identify days with air pollution due to the smoke haze allows us to include two additional years of data in our analysis. Based on the level of  $PM_{10}$  chosen for the above analysis, we determined the corresponding cut-off for visibility of approximately 1.1 km. This yields a total of 14 low-visibility days associated with the smoke

haze for the period 1994-97. Note that the values of  $PM_{10}$  and inverse visibility are highly correlated ( $\rho$ = .83).

With the longer window for studying the relationship between the smoke haze and mortality, several new findings emerge (see Table 5). First, the risk of death for infants is significantly higher following a high air pollution day for total, non-traumatic, and "other" mortality. For non-traumatic infant mortality, a high air pollution day is associated with a 64% increase in the risk of death and this effect is significant at the five-percent level. There are too few infant deaths due to cardiovascular or respiratory causes to model these outcomes. The results for infants are somewhat sensitive to selecting a lower cut-off when classifying days as having high levels of air pollution. In particular, reducing the cut-off moderately can reduce the magnitude of these mortality effects and their statistical significance. Second, mortality effects for the all-age and elderly (ages 65-74) groups are quantitatively and qualitatively similar to those based on the  $PM_{10}$  measures. With these data it is possible to estimate models for deaths due to cardiovascular and respiratory causes, although the caveats regarding the unassigned causes of death continue to hold. The risk of death due to cardiovascular causes for individuals aged 65-74 is twice as high on days following a smoke haze episode, and this effect is significant at the one-percent level. Positive but insignificant effects for mortality due to respiratory causes emerge for individuals aged 65-74 and 75 plus and for mortality due to cardiovascular and respiratory causes for individuals of all ages.

For both the  $PM_{10}$  and visibility models, deaths due to "other" non-traumatic causes (i.e., excluding cardiovascular or respiratory mortality) are significantly higher following a high air pollution day. This holds for models based on all-age mortality and mortality among individuals aged 65-74 years; for infants, a significant effect emerges only in the visibility-based models. We explored the specific causes of death that underlie these significant effects (results not shown). The causes of death that emerged as important were chronic conditions such as genitourinary causes (especially kidney disease) and neoplasms; perinatal causes were important for infants.

#### Visibility Models for Other Malaysian Cities

We next turn to the effects of the smoke haze on daily mortality for major Malaysian cities other than Kuala Lumpur. The analysis for Kuala Lumpur allowed us to identify the threshold below which low visibility due to smoke haze may be associated with higher daily mortality, calibrated on the basis of corresponding PM<sub>10</sub> levels. Using this same visibility threshold, we examine the effects of smoke haze on daily mortality in four other major cities in Malaysia. Table 6 shows the mean number of daily deaths and the total number of days with visibility less than 1.1 kilometers for Kuala Lumpur and the four other cities: Johore Bahru, Ipoh, Kuching, and Penang. Kuching is located in Sarawak, on the Malaysian portion of Borneo; Ipoh is in central Peninsular Malaysia, north of Kuala Lumpur; Johore Bahru is on the southern tip of Peninsular Malaysian, across a narrow strait from Singapore; and, finally, Penang is an island off the coast of northwest Peninsular Malaysia. Although Kuching has the lowest mean daily deaths of this group, it has the largest number of low visibility days. Note that Kuching, a city of approximately 400,000 people, is located directly downwind from the Indonesian province of Kalimantan, one area where the forest fires were concentrated.

Other than Kuala Lumpur, Kuching is the only city for which there emerged significant effects of the smoke haze on mortality. There are many reasons, apart from small number of

low-visibility days, why significant mortality effects were not apparent in the other cities. One is that visibility is not an ideal indicator: especially when compared to  $PM_{10}$ , it is imprecise and is subject to uncertain measurement error. However, even with a better measure of air pollution, the topography and physical layout of each city may affect the relationship between air pollution and mortality in unknown ways. Finally, the sources of emissions may determine the effects of air pollution on mortality. That is, with the same visibility level, there may be poorer air quality in Kuala Lumpur because it is comprised to a greater extent of emissions from motor vehicles and industry.

Tables 7 and 8 show summary statistics for daily mortality and for weather and air quality in Kuching over the period 1994-97. In Table 9 we present regression results that show the effects of the smoke haze on daily mortality. Three interesting patterns emerge. First, high air pollution days are associated with significantly higher mortality due to cardiovascular and respiratory causes (at the ten-percent significance level) for all ages combined. Mortality due to cardiovascular causes is 1.5 times higher immediately following a high air pollution day and respiratory mortality is 2.4 times higher. Second, there is a consistent pattern across age groups of exposure to smoke haze being associated with increased respiratory deaths. For each age group other than infants, high air pollution days are associated with higher mortality due to respiratory causes, although the effects are significant, at the ten-percent level, only for age groups 65-74 and 75 and older. Third, mortality effects associated with a high air pollution day are concentrated in the oldest age group, comprised of individuals aged 75 and older. For this group there are significant effects—at the one-percent level—for total mortality and deaths due to non-external causes. These effects are driven in large part by the substantially higher risk of death due to cardiovascular causes. A high air pollution day is associated with a 3-fold increase in cardiovascular-related mortality. Mortality effects for deaths due to respiratory and other causes are positive and fairly large, although only the former effect is significant (at the tenpercent level).

There is a notably different mortality pattern associated with high air pollution days in Kuching compared to Kuala Lumpur. In Kuala Lumpur the effects are concentrated in the age group 65-74, while in Kuching they are greatest for individuals aged 75 and over. Several possible factors may account for these differences, including the severity of pollution, the cause of death structure, data quality, and differential behavioral responses. The simplest explanation, however, is that while the effects of the smoke were much worse in Kuching, the remainder of the time air quality there is much better than in Kuala Lumpur. Thus, those susceptible to air pollution effects are likely to die at earlier ages in Kuala Lumpur, with only those who are more robust surviving to ages 75 and older. In Kuching, on the other hand, people susceptible to the effects of air pollution generally live longer—because of the better air quality—but an episode of high air pollution takes a large toll. This interpretation is supported by the analysis of lagged effects, discussed below.

#### Autoregressive Models

The results discussed above are based on standard models that do not account for the possible autoregressive nature of the data. An autoregressive structure may arise because of either positive or negative correlation among the number of deaths on neighboring days. Positive correlation may be caused by neighboring days having similar levels of smoke haze, weather, and other causal factors. Negative correlation, on the other hand, may be the result of

harvesting-type effects, whereby high mortality on one day is balanced by lower mortality on subsequent days—in essence, a positive mortality shock is recouped over time.

In order to investigate the effects of autocorrelation on our results we reestimated the models using a range of low-order autoregressive specifications. We estimated models with autoregression of order 1, 2, and 3. These results are presented in Table 10. This table, and our discussion below, focuses exclusively on results for models with statistically significant effects (at the five-percent level) in the standard regression models. There are three panels in Table 10. The first panel shows results for Kuala Lumpur based on the PM<sub>10</sub> models; the second and third panels show results of the visibility models for Kuala Lumpur and Kuching, respectively.

For the PM<sub>10</sub> models for Kuala Lumpur, the effects of a high air pollution day are attenuated only slightly—if at all—with controls for autocorrelation. The level of statistical significance is in all but one case the same as in the corresponding standard model. Not shown in the table are the autocorrelation parameters. Virtually all of these are negative in sign and small in magnitude, falling in the range between -0.10 and 0. This negative autocorrelation indicates that there is some displacement of mortality among neighboring days due to harvesting-type effects. However, it does not provide an estimate of the magnitude of this displacement, because the autocorrelation parameters also reflect offsetting effects due to positive autocorrelation in weather and air pollution, which are high. First-order autocorrelation for PM<sub>10</sub> is 0.8 and for temperature is 0.5. In the visibility models, controlling for the autoregressive structure has similar effects on the estimated parameters of interest. In particular, when the effects of a high air pollution day are attenuated, they are only slightly lower than in the corresponding standard model. In many cases, however, the standard errors are actually smaller in models that account for autocorrelation.

In summary, the modest degree of autocorrelation in the daily mortality series for both Kuala Lumpur and Kuching means that estimating autoregressive models does not alter qualitatively or quantitatively the results described in the previous section based on standard regression models.

#### Lagged Effects

To this point, the focus of our analysis has been the one-day lagged mortality effects of high air pollution levels, which represents the immediate consequences of exposure to the smoke haze. In order to investigate the possible extent to which these effects represent a longer-lasting mortality shock we estimated models that included an extended series of lagged effects. The results of this exercise are summarized in Table 11, which shows the longest cumulative lag that is positive and statistically significant at the five-percent level for each mortality outcome with a statistically significant short-term effect. Note that we are examining the sign and statistical significance of the sum of the coefficients for the series of estimated lagged effects. The coefficients for the lags that are being summed are both positive and negative—generally, they alternate between positive and negative although no fixed pattern is apparent.

The results in Table 11 indicate that with the exception of mortality among the elderly aged 65-74 in Kuala Lumpur, the effects of a high air pollution day are very short-lived. In particular, higher mortality one day after a smoke haze episode is offset by lower mortality on the following day, two days after the initial episode of high air pollution. This is the result for all-age and infant mortality in Kuala Lumpur and for elderly (aged 75+) mortality in Kuching. However, for the elderly aged 65-74 in Kuala Lumpur, increases in mortality resulting from a

high air pollution day are more persistent. Focusing on total deaths due to non-traumatic causes, the cumulative mortality effect is positive for a nine-day period in the  $PM_{10}$  models and for a five-day period in the visibility models. It is important to note, moreover, that effects are positive and large over longer periods, although the cumulative effects are not significant. The results in Table 12 illustrate this point. The cumulative lagged effects are large—and statistically significant at the ten-percent level—over a period of almost three weeks following a high air pollution day.

#### VII. Conclusions

The 1997 forest fires in Southeast Asia were an environmental disaster of huge proportions, in terms of their intensity, extent, duration, and the number of people affected (Nichol, 1998; Brauer and Hisham-Hashim, 1998; Economist, 1997b). In this study, we overcame many of the limitations of previous studies on this topic to show that the smoke haze from these fires had a deleterious effect on population health in Malaysia.

A major question regarding the health impact concerns the extent to which the increased mortality simply represented a "harvesting" effect, in which the observed deaths were displaced a few days. Our results indicate that the displacement of deaths due to the smoke haze was very short-term. However, for one segment of the Malaysian population—elderly aged 65-74 in Kuala Lumpur—there was an upward shift in mortality lasting at least a few weeks. The overall mortality burden, in terms of days of healthy life lost, is nevertheless likely to be quite small. However, the mortality measures examined here serve as sentinel indicators and are thus suggestive of wider short-term health impacts—particularly with respect to acute morbidity. It is difficult though to assess the precise magnitude and character of these health impacts without data on morbidity from an unbiased and representative sample, which are unavailable. Regardless of the short-term mortality effects of high air pollution, there may be long-term health effects associated with exposure to elevated levels of air pollution over an extended period. There is a clear need to develop a better understanding of the long-term effects of air pollution in various settings around the world.

One implication of our results from studying the short-term effects in Malaysia of the smoke haze is that the effects in Indonesia itself must have been very large. The presence of significant mortality effects in Malaysian cities that are several hundred miles away from the main fires strongly supports this notion. Unfortunately, there are no appropriate health or mortality data available for Indonesia to study this issue directly. It is however possible to construct visibility-based indicators of severe smoke haze (defined the same as for Malaysia) for various Indonesian cities, although missing values are a problem. Among the stations with the least missing data, there were at least 57 low-visibility days in 1997 in Sibolga, a city in northwest Sumatra; 102 days in Rengat in Central Sumatra; and 145 days in Palangaraya in South-Central Kalimantan. This compares to a total of 14 high smoke haze days in Kuala Lumpur and 33 in Kuching that same year.

President Suharto of Indonesia apologized for the forest fires and designated their effects a "natural disaster" caused by the El Niño weather phenomenon. This was disingenuous given the well-known causes of the forest fires. To prevent a recurrence of the forest fires in Indonesia it is especially important that the existing laws against burning need are enforced. This involves better public education concerning the laws as well as the causes and consequences of the fires.

Also needed are better monitoring of forest fires and the imposition of real punishments on those who break this law.

There is also a clear need for changes to certain existing policies and programs in Indonesia. For instance, participants in the Transmigration Program should be trained in new farming techniques to better exploit peat soils. At a more fundamental level, property-rights need to be better defined since ill-defined rights lead to conflicts between locals, migrants, and forestry firms, with fire used as a weapon by all sides (Economist, 1998). Although the best solution is to avoid fires in first place, there have to be better response plans for dealing with fires when they do occur. There are several possible strategies for controlling and extinguishing fires. These must rely on local forest fire fighting capabilities, which should be developed. Changes in population and forestry concession policies may also be necessary. Population policies clearly play an important role, especially in the long term. Foremost is the Transmigration Project that encourages millions of people to move from crowded islands of Java and Bali to less densely populated but heavily forested islands such as Kalimantan, Sumatra, and Irian Jaya. Current policies on forestry concessions encourage short-term exploitation rather than sustainability and should be changed. Underlying all of these policy changes is a need to promote rural development in Indonesia.

More assertiveness on the part of Indonesia's neighbors is warranted, given that the fallout from the fires clearly affects them. In particular, the transboundary incidence of adverse economic effects (see Glover and Jessup, 1999) and health effects (from this study) is now clear. Finally, given the global consequences of destruction of a massive area of rainforest, huge emission of greenhouse gases, and future global warming, there ought to be a wider international response as well.

This last point is especially important because the 1997 forest fire and smoke haze episode was not an isolated event. Large forest fires occurred in other locations around the globe (though with a smaller affected population) and in previous and subsequent years in Southeast Asia. In particular, forest fires raged again in the region in 1998 and 2000 (Economist, 1998 and 2000). In Indonesia, the poor record on enforcement of existing policies and the absence of new policy initiatives has now been combined with major—and continuing—economic and political upheaval. This suggests that recent disasters will probably be repeated. However, successful efforts to prevent and contain forest fires will have important economic and health benefits.

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Table 1. Daily Mortality in Kuala Lumpur, 1994-97

					Percentile			
	Mean (SD)	Min	10th	25th	50th	75th	90th	Max
Total	16.1 (4.3)	3	11	13	16	19	22	35
Non-traumatic	14.7 (4.1)	3	9	12	15	17	20	33
Cardiovascular	4.8 (2.3)	0	2	3	5	6	8	14
Respiratory	0.9(0.9)	0	0	0	1	1	2	5
Other	8.9 (3.1)	0	5	7	9	11	13	25
Age < 1	1.1 (1.1)	0	0	0	1	2	3	7
Age 1-64	7.5 (2.9)	1	4	5	7	9	11	18
Age 65-74	3.1 (1.7)	0	1	2	3	4	5	9
Age > 75	4.2 (2.1)	0	2	3	4	6	7	12

Source: Malaysian vital statistics.

Table 2. Climate and Air Quality in Kuala Lumpur, 1996-97

					Percentile			
	Mean (SD)	Min	10th	25th	50th	75th	90th	Max
Temperature Mean	81.6 (2.0)	75.3	79.0	80.2	81.6	83.0	84.1	87.3
Mean Dew Point	74.1 (1.3)	67.7	72.5	73.5	74.2	75.0	75.5	77.2
Particulates								
$PM_{10}$	64.2 (43.0)	16.2	33.3	41.1	54.6	71.0	99.4	423.9
Visibility	6.8 (2.4)	0.3	3.8	5.5	6.9	8.3	9.9	15.3

Note: Temperatures are measured in Fahrenheit,  $PM_{10}$  in  $\mu g/m^3$ , and visibility in kilometers. Sources: See text.

Table 3. Effects of Continuous and Discrete Measures of One-Day Lagged  $PM_{10}$  on Non-Traumatic Deaths Ages 65-74, Kuala Lumpur, 1996-97

	PM <sub>10</sub> Measure		
	Continuous	Discrete	
Beta	0.00134	0.540	
S.E.	0.000754	0.238	
Z-statistic	1.773	2.274	
Relative risk of high pollution day <sup>1</sup>	$1.39^{2}$	1.72	

#### Notes:

- 1. A high air pollution day is defined as having  $PM_{10} > 210\mu g/m^3$ .
- 2. Mean PM<sub>10</sub> is 60.60 on low air pollution days and 305.93 on high air pollution days for a difference of 245.33. This leads to an effect of  $\exp(245.33 \times 0.00134) = 1.39$

Table 4. Adjusted relative risk of mortality following a high air pollution day ( $PM_{10} > 210 \ \mu g/m^3$ ) for Kuala Lumpur, Malaysia, 1996-97

Age Group	Total	Non-Traumatic	Cardiovascular	Respiratory	Other
All	1.221** (0.118)	1.214* (0.126)	-	-	1.298* (0.180)
< 1	1.039 (0.315)	0.988 (0.322)	-	. <del>-</del>	0.969 (0.324)
1-64	1.099 (0.153)	1.042 (0.160)			1.142 (0.248)
65-74	1.571** (0.360)	1.716** (0.408)	-	-	2.423*** (0.846)
75+	1.303 (0.274)	1.303 (0.275)	-	-	1.209 (0.369)

Notes: Standard errors in parentheses; \* p<.10; \*\* p<.05; \*\*\*p<.01.

Models control for weather conditions (temperature and humidity), a time trend (capturing changes in population and mortality), and overdispersion.

Table 5. Adjusted relative risk of mortality following a high air pollution day (visibility < 1.1 km) for Kuala Lumpur, Malaysia, 1994-97

		Mortality Measure							
Age Group	Total	Non-Traumatic	Cardiovascular	Respiratory	Other				
All	1.226***	1.234***	1.235	1.390	1.218**				
	(0.090)	(0.096)	(0.170)	(0.434)	(0.122)				
< 1	1.665** (0.386)	1.636** (0.411)	-	-	1.589* (0.413)				
1-64	1.114	1.123	1.119	0.784	1.172				
	(0.120)	(0.133)	(0.230)	(0.376)	(0.188)				
65-74	1.697***	1.752***	2.020***	1.946	1.553*				
	(0.273)	(0.286)	(0.523)	(0.938)	(0.362)				
75+	0.932	0.916	0.890	1.689	0.872				
	(0.156)	(0.155)	(0.279)	(0.861)	(0.194)				

Notes: Standard errors in parentheses; \* p < .10; \*\* p < .05; \*\*\*p < .01.

Models control for weather conditions (temperature and humidity), a time trend (capturing changes in population and mortality), and overdispersion.

Table 6. Mortality and Air Pollution in Selected Malaysian Cities, 1994-97

City	Mean Daily Deaths	Days with Visibility < 1.1 km
Johore Bahru	5.7	2
Ipoh	7.1	12
Kuching	2.5	33
Penang	4.7	8
Kuala Lumpur	16.1	14

Sources: See text.

Table 7. Daily Mortality in Kuching, 1994-97

					Percentile			
	Mean (SD)	Min	10 <sup>th</sup>	25th	50th	75 <sup>th</sup>	90th	Max
Total	2.5 (1.6)	0	1	1	2	3	5	9
Non-traumatic	2.3 (1.6)	0	0	1	2	3	4	9
Cardiovascular	0.9 (0.9)	0	0	0	1	1	2	5
Respiratory	0.2 (0.4)	0	0	0	0	0	1	3
Other	1.3 (1.1)	0	0	1	1	2	3	6
Age < 1	0.2 (0.5)	0	0	0	0	0	1	3
Age 1-64	1.5 (1.2)	0	0	1	1	2	3	8
Age 65-74	0.5 (0.7)	0	0	0	0	1	1	3
Age > 75	0.4 (0.6)	0	0	0	0	1	11	4

Source: Malaysian vital statistics.

Table 8. Climate and Air Quality in Kuching, 1994-97

		,			Percentile			
	Mean (SD)	Min	10th	25th	50th	75th	90th	Max
Temperature Mean Mean Dew Point	79.2 (2.0) 74.2 (1.1)	73.3 68.6	76.8 72.9	77.8 73.6	79.2 74.2	80.6 74.9	81.9 75.6	85.1 77.4
Visibility	10.5 (3.5)	0.1	5.0	9.0	11.4	12.9	14.1	17.2

Notes: Temperatures are measured in Fahrenheit and visibility in kilometers.

Sources: See text.

Table 9. Adjusted relative risk of mortality following a high air pollution day (visibility < 1.1 km) for Kuching, Malaysia, 1994-97

	Mortality Measure								
Age Group	Total	Non-Traumatic	Cardiovascular	Respiratory	Other				
Ali	1.160	1.120	1.548*	2.399*	0.775				
	(0.160)	(0.171)	(0.396)	(1.079)	(0.143)				
< 1	0.853 (0.369)	0.853 (0.319)	-	-	1.075 (0.404)				
1-64	1.019	0.981	1.636	1.777	0.595*				
	(0.169)	(0.214)	(0.517)	(0.681)	(0.158)				
65-74	0.840	0.836	0.985	2.646*	0.618				
	(0.278)	(0.275)	(0.506)	(1.503)	(0.254)				
75+	2.387***	2.568***	3.121***	2.363*	1.835				
	(0.757)	(0.832)	(1.389)	(1.148)	(0.717)				

Notes: Standard errors in parentheses; \* p < .10; \*\* p < .05; \*\*\*p < .01.

Models control for weather conditions (temperature and humidity), a time trend (capturing changes in population and mortality), and overdispersion.

Table 10. Autoregressive Models for Daily Mortality Effects of High Air Pollution

New	Age	Mortality	Std.	AR(1)	AR(2)	AR(3)		
All         Total         1.221** (0.118) (0.111) (0.112) (0.110)           65-74         Total         1.571** (0.360) (0.335) (0.323) (0.333) (0.333)           65-74         Non- 1.716** 1.697** 1.701** 1.719**           65-74         Non- Traumatic (0.408) (0.375) (0.374) (0.375) (0.374) (0.375)           65-74         Other (0.846) (0.777) (0.748) (0.741)           Kuala Lumpur Visibility           All         Total (0.090) (0.088) (0.089) (0.086) (0.089) (0.086)           All         Non- 1.234*** 1.225*** 1.228*** 1.228*** 1.228*** (0.120)* (0.093) (0.095) (0.093)           All         Other (0.122) (0.117) (0.119) (0.116)           <1		Measure						
All Total (0.118) (0.111) (0.112) (0.110)  65-74 Total (0.360) (0.335) (0.323) (0.333)  65-74 Non- 1.716** 1.697** 1.701** 1.719**  65-74 Other (0.408) (0.375) (0.374) (0.375)  65-74 Other (0.846) (0.777) (0.748) (0.741)  **Traumatic (0.408) (0.375) (0.374) (0.375)***  65-74 Other (0.846) (0.777) (0.748) (0.741)  **Traumatic (0.846) (0.777) (0.748) (0.741)  **All Total (0.090) (0.088) (0.089) (0.086)  All Non- 1.234*** 1.225*** 1.228*** 1.228***  61 Total (0.096) (0.093) (0.095) (0.093)  All Other (0.122) (0.117) (0.119) (0.116)  **Traumatic (0.386) (0.380) (0.379) (0.379)  **Traumatic (0.411) (0.376) (0.375) (0.375)  65-74 Total (0.273) (0.253) (0.251) (0.248)  **Traumatic (0.286) (0.262) (0.259) (0.258)  **Traumatic (0.286) (0.262) (0.259) (0.258)  **Traumatic (0.523) (0.468) (0.466) (0.465)  **Traumatic (0.523) (0.468) (0.466) (0.465)  **Traumatic (0.647) (0.649) (0.658) (0.684)  **Total (0.757) (0.760) (0.748) (0.735)  **Traumatic (0.832) (0.833) (0.816) (0.799)					1 101*	1 185*		
Total	All	Total						
Total								
Non-	65-74	Total						
65-74         Traumatic         (0.408)         (0.375)         (0.374)         (0.375)           65-74         Other         2.423***         2.406***         2.375***         2.375***           Kuala Lumpur Visibility           Kuala Lumpur Visibility           All         Total         (0.090)         (0.088)         (0.089)         (0.086)           All         Non-         1.234***         1.225***         1.228***         1.228***           All         Other         (0.096)         (0.093)         (0.095)         (0.093)           All         Other         (0.096)         (0.093)         (0.095)         (0.093)           All         Other         (0.122)         (0.117)         (0.119)         (0.116)           <1		Man				•		
65-74         Other         2.423*** (0.846) (0.777) (0.748) (0.741)           Kuala Lumpur Visibility           All         Total (0.090) (0.088) (0.089) (0.086) (0.089) (0.086)           All Non- Traumatic (0.096) (0.093) (0.095) (0.093) (0.095) (0.093)           All Other (0.122) (0.117) (0.119) (0.116)           < 1         Total (0.386) (0.380) (0.379) (0.379)           < 1         Non- (0.386) (0.380) (0.379) (0.379)           < 1         Non- (0.411) (0.376) (0.375) (0.375)           < 1         Total (0.273) (0.253) (0.251) (0.248)           < 1         Traumatic (0.286) (0.262) (0.259) (0.258)           < 2-74 Cardio- vascular (0.523) (0.468) (0.466) (0.465)           Kuching Visibility           All Respiratory         2.048** (2.048** (2.042*** (2.065** (2.067** (0.647) (0.649) (0.658) (0.684)           < 1         Total (0.757) (0.760) (0.748) (0.735)           < 2.568*** (2.540*** (2.542*** (2.529*** (2.529*** (2.529*** (0.759) (0.759) (0.759) (0.755)           < 2.568*** (0.832) (0.833) (0.816) (0.799)           < 2.54         Cardio- (0.832) (0.833) (0.816) (0.799)           < 2.54         Cardio- (0.832) (0.833) (0.816) (0.799)	65-74							
Kuala Lumpur Visibility           Kuala Lumpur Visibility           All         Total         1.226*** 1.218*** 1.219*** 1.218*** 1.228*** 1.228*** 1.228*** 1.228*** 1.228*** 1.228*** 1.228*** 1.228*** 1.228*** 1.228*** 1.228*** 1.228*** 1.208* 1.210* 1.203* (0.095) (0.093)           All         Other         1.218** 1.208* 1.210* 1.203* (0.117) (0.119) (0.116)           < 1         Total (0.386) (0.380) (0.379) (0.379) (0.379)           < 1         Non- (0.386) (0.380) (0.379) (0.379) (0.379)           < 1         Non- (0.411) (0.376) (0.375) (0.375)           65-74         Total (0.273) (0.253) (0.251) (0.248)           65-74         Non- (0.286) (0.262) (0.259) (0.258)           65-74         Cardio- (0.286) (0.262) (0.259) (0.258)           Kuching Visibility           All           Respiratory         2.048** (2.048** (2.042*** (2.065** (2.067** (0.647) (0.649) (0.658) (0.684) (0.465)           Total (0.757) (0.760) (0.748) (0.735)           Non- (0.528) (0.757) (0.760) (0.748) (0.735)           Non- (0.757) (0.760) (0.748) (0.735)           Non- (0.757) (0.760) (0.816) (0.799)           75+ (0.760) (0.832) (0.833) (0.816) (0.799)           75+ (0.760) (0.748) (0.735)           Total (0.832) (0.833) (0.816) (0.799)		Haumanc						
$\begin{array}{c ccccccccccccccccccccccccccccccccccc$	65-74	Other						
All Total 1.226*** 1.218*** 1.219*** 1.218***    Non-					(0.740)	(0.741)		
All Total (0.090) (0.088) (0.089) (0.086)  All Non- 1.234*** 1.225*** 1.228*** 1.228***  All Other (0.096) (0.093) (0.095) (0.093)  All Other 1.218** 1.208* 1.210* 1.203*  (0.122) (0.117) (0.119) (0.116)  (1.665** 1.652** 1.675** 1.677**  (0.386) (0.380) (0.379) (0.379)  (0.379)  All Non- 1.636** 1.619** 1.644** 1.644**  Traumatic (0.411) (0.376) (0.375) (0.375)  (0.273) (0.253) (0.251) (0.248)  All Non- 1.752*** 1.737*** 1.737*** 1.740***  Traumatic (0.286) (0.262) (0.259) (0.258)  Cardio- 2.020*** 2.016*** 2.042*** 2.096***  Vascular (0.523) (0.468) (0.466) (0.465)  Kuching Visibility  All Respiratory (0.647) (0.649) (0.658) (0.684)  75+ Total (0.757) (0.760) (0.748) (0.735)  Traumatic (0.832) (0.833) (0.816) (0.799)  Traumatic (0.832) (0.833) (0.816) (0.799)  Cardio- 3.121*** 3.059** 3.117**		K			4.646444	4 0 4 O de de de		
All Non- Traumatic (0.096) (0.093) (0.095) (0.093)  All Other (0.122) (0.117) (0.119) (0.116)  <1 Total (0.386) (0.380) (0.379) (0.379)  \[ \begin{array}{c ccccccccccccccccccccccccccccccccccc	A11	Total						
All Traumatic (0.096) (0.093) (0.095) (0.093)  All Other (0.122) (0.117) (0.119) (0.116)  <1 Total (0.386) (0.380) (0.379) (0.379)  <1 Non- (0.411) (0.376) (0.375) (0.375)  All Total (0.411) (0.376) (0.375) (0.375)  All Total (0.273) (0.253) (0.251) (0.248)  All Non- (0.286) (0.262) (0.259) (0.258)  Cardio- (0.523) (0.468) (0.466) (0.465)   Kuching Visibility  All Respiratory (0.647) (0.649) (0.658) (0.684)  75+ Total (0.757) (0.760) (0.748) (0.735)  All Non- (0.523) (0.833) (0.816) (0.799)  Traumatic (0.832) (0.833) (0.816) (0.799)  Cardio- (0.757) (0.769) (0.816) (0.799)  Cardio- (0.832) (0.833) (0.816) (0.799)	4 111	1000	•	,	,			
All Other 1.218** 1.208* 1.210* 1.203* (0.122) (0.117) (0.119) (0.116)  <1 Total 1.665** 1.652** 1.675** 1.677** (0.386) (0.380) (0.379) (0.379)  <1 Non- 1.636** 1.619** 1.644** 1.644** (0.411) (0.376) (0.375) (0.375)  65-74 Total 1.697*** 1.685*** 1.687*** 1.690*** (0.273) (0.253) (0.251) (0.248)  65-74 Non- 1.752*** 1.737*** 1.737*** 1.740*** (0.286) (0.262) (0.259) (0.258)  Cardio- 2.020*** 2.016*** 2.042*** 2.096*** (0.523) (0.468) (0.466) (0.465)   Kuching Visibility  All Respiratory 2.048** 2.048** 2.065** 2.067** (0.647) (0.649) (0.658) (0.684)  75+ Total (0.757) (0.760) (0.748) (0.735) (0.759) (0.832) (0.833) (0.816) (0.799)  Traumatic (0.832) (0.833) (0.816) (0.799)  Cardio- 3.121*** 3.059** 3.117** 3.117**	A 11	Non-						
All       Other       (0.122)       (0.117)       (0.119)       (0.116)         <1	All	Traumatic	•	•	,			
Total   1.665**   1.652**   1.675**   1.677**   (0.386)   (0.380)   (0.379)   (0.379)   (0.379)   (0.379)   (0.379)   (0.379)   (0.379)   (0.379)   (0.379)   (0.379)   (0.379)   (0.379)   (0.379)   (0.379)   (0.375)   (0.375)   (0.375)   (0.375)   (0.375)   (0.375)   (0.375)   (0.375)   (0.375)   (0.273)   (0.253)   (0.251)   (0.248)   (0.273)   (0.253)   (0.251)   (0.248)   (0.273)   (0.253)   (0.251)   (0.248)   (0.262)   (0.259)   (0.258)   (0.259)   (0.259)   (0.258)   (0.259)   (0.258)   (0.259)   (0.259)   (0.258)   (0.259)   (0.259)   (0.258)   (0.259)   (0.259)   (0.258)   (0.259)   (0.259)   (0.258)   (0.259)   (0.259)   (0.259)   (0.259)   (0.259)   (0.259)   (0.259)   (0.259)   (0.259)   (0.259)   (0.259)   (0.259)   (0.259)   (0.259)   (0.259)   (0.259)   (0.259)   (0.259)   (0.259	A 13	Othor	1.218**	1.208*				
<1         Total         (0.386)         (0.380)         (0.379)         (0.379)           <1	All	Other	(0.122)	(0.117)	(0.119)	(0.116)		
Non-	. 1	T-4-1	1.665**	1.652**	1.675**	1.677**		
Traumatic (0.411) (0.376) (0.375) (0.375)  1.697*** 1.685*** 1.687*** 1.690*** (0.273) (0.253) (0.251) (0.248)  Non- 1.752*** 1.737*** 1.737*** 1.740***  Traumatic (0.286) (0.262) (0.259) (0.258)  Cardio- 2.020*** 2.016*** 2.042*** 2.096*** vascular (0.523) (0.468) (0.466) (0.465)  Kuching Visibility  All Respiratory 2.048** 2.048** 2.065** 2.067** (0.647) (0.649) (0.658) (0.684)  75+ Total 2.387*** 2.382*** 2.389*** 2.387*** (0.757) (0.760) (0.748) (0.735)  Non- 2.568*** 2.540*** 2.542*** 2.529*** Traumatic (0.832) (0.833) (0.816) (0.799)  Cardio- 3.121*** 3.059** 3.117** 3.117**	< 1	1 otai	(0.386)	(0.380)	(0.379)			
Traumatic   (0.411)   (0.376)   (0.373)   (0.373)   (0.373)   (0.373)   (0.373)   (0.373)   (0.273)   (0.253)   (0.251)   (0.248)   (0.273)   (0.253)   (0.251)   (0.248)   (0.265)   (0.258)   (0.258)   (0.262)   (0.259)   (0.258)   (0.265)   (0.262)   (0.259)   (0.258)   (0.523)   (0.468)   (0.466)   (0.465)   (0.465)		Non-	1.636**	1.619**	1.644**	1.644**		
65-74 Total (0.273) (0.253) (0.251) (0.248)  Non- 1.752*** 1.737*** 1.737*** 1.740***  (0.286) (0.262) (0.259) (0.258)  Cardio- 2.020*** 2.016*** 2.042*** 2.096***  vascular (0.523) (0.468) (0.466) (0.465)   Kuching Visibility  All Respiratory 2.048** 2.048** 2.065** 2.067** (0.647) (0.649) (0.658) (0.684)  75+ Total 2.387*** 2.382*** 2.389*** 2.387*** (0.757) (0.760) (0.748) (0.735)  Non- 2.568*** 2.540*** 2.542*** 2.529***  Traumatic (0.832) (0.833) (0.816) (0.799)  Cardio- 3.121*** 3.059** 3.117** 3.117**	< 1	Traumatic	(0.411)	(0.376)	(0.375)	(0.375)		
Non-   1.752***   1.737***   1.737***   1.740***		T . 1	1.697***	1.685***	1.687***	1.690***		
65-74         Traumatic Cardio-vascular         (0.286)         (0.262)         (0.259)         (0.258)           Example 10.523         (0.462)         (0.259)         (0.258)           Example 2.020*** 2.016*** 2.042*** 2.096***           Vascular (0.523)         (0.468)         (0.466)         (0.465)           Example 2.048** 2.048** 2.065** 2.067**           (0.647)         (0.649)         (0.658)         (0.684)           75+         Total 2.387*** 2.382*** 2.389*** 2.387***         (0.735)           75+         Non- 2.568*** 2.540*** 2.542*** 2.529***           75+         Traumatic (0.832)         (0.833)         (0.816)         (0.799)           75+         Cardio- 3.121*** 3.059** 3.117** 3.117**	65-74	Total	(0.273)	(0.253)	(0.251)	(0.248)		
Traumatic (0.286) (0.262) (0.259) (0.258)  Cardio- vascular (0.523) (0.468) (0.466) (0.465)  Kuching Visibility  All Respiratory (0.647) (0.649) (0.658) (0.684)  Total (0.757) (0.760) (0.748) (0.735)  Non- Traumatic (0.832) (0.833) (0.816) (0.799)  Cardio-  Cardio		Non-	1.752***	1.737***	1.737***	1.740***		
Vascular         (0.523)         (0.468)         (0.466)         (0.465)           Kuching Visibility           All         Respiratory         2.048**         2.048**         2.065**         2.067**           (0.647)         (0.649)         (0.658)         (0.684)           75+         Total         2.387***         2.382***         2.389***         2.387***           75+         Non-         2.568***         2.540***         2.542***         2.529***           75+         Traumatic         (0.832)         (0.833)         (0.816)         (0.799)           75+         Cardio-         3.121***         3.059**         3.117**         3.117**	65-74		(0.286)	(0.262)	(0.259)	(0.258)		
Vascular         (0.523)         (0.468)         (0.466)         (0.465)           Kuching Visibility           All         Respiratory         2.048**         2.048**         2.065**         2.067**           (0.647)         (0.649)         (0.658)         (0.684)           75+         Total         2.387***         2.382***         2.389***         2.387***           75+         Non-         2.568***         2.540***         2.542***         2.529***           75+         Traumatic         (0.832)         (0.833)         (0.816)         (0.799)           75+         Cardio-         3.121***         3.059**         3.117**         3.117**		Cardio-	2.020***	2.016***	2.042***	2.096***		
Kuching VisibilityAllRespiratory $2.048**$ $2.048**$ $2.065**$ $2.067**$ 75+Total $2.387***$ $2.382***$ $2.389***$ $2.387***$ 75+Non- Traumatic $2.568***$ $2.540***$ $2.542***$ $2.529***$ 75+Cardio- $3.121***$ $3.059**$ $3.117**$ $3.117**$	65-74			(0.468)	(0.466)	(0.465)		
All Respiratory 2.048** 2.048** 2.065** 2.067** (0.647) (0.649) (0.658) (0.684)  75+ Total 2.387*** 2.382*** 2.389*** 2.387*** (0.757) (0.760) (0.748) (0.735)  75+ Non- 2.568*** 2.540*** 2.542*** 2.529*** Traumatic (0.832) (0.833) (0.816) (0.799)  75+ Cardio- 3.121*** 3.059** 3.117**			Kuching V	isibility				
All Respiratory (0.647) (0.649) (0.658) (0.684)  75+ Total 2.387*** 2.382*** 2.389*** 2.387*** (0.757) (0.760) (0.748) (0.735)  75+ Non- 2.568*** 2.540*** 2.542*** 2.529*** Traumatic (0.832) (0.833) (0.816) (0.799)  Cardio- 3.121*** 3.059** 3.117** 3.117**					2.065**	2.067**		
75+ Total 2.387*** 2.382*** 2.389*** 2.387*** (0.757) (0.760) (0.748) (0.735)  75+ Non- 2.568*** 2.540*** 2.542*** 2.529*** Traumatic (0.832) (0.833) (0.816) (0.799)  75+ Cardio- 3.121*** 3.059** 3.117**	All	Respiratory						
75+ Total (0.757) (0.760) (0.748) (0.735)  Non- 2.568*** 2.540*** 2.542*** 2.529***  Traumatic (0.832) (0.833) (0.816) (0.799)  Cardio- 3.121*** 3.059** 3.117**						2.387***		
75+ Non- 2.568*** 2.540*** 2.542*** 2.529*** Traumatic (0.832) (0.833) (0.816) (0.799) Cardio- 3.121*** 3.059** 3.117**	75+	Total						
75+ Traumatic (0.832) (0.833) (0.816) (0.799)  Cardio- 3.121*** 3.059** 3.117** 3.117**		Non-	,	,	,			
Cardio- 3.121*** 3.059** 3.117** 3.117**	75+							
75±					,			
	75+							

Notes: Standard errors in parentheses; \* p < .10; \*\* p < .05; \*\*\*p < .01.

Models control for weather conditions (temperature and humidity), a time trend (capturing changes in population and mortality), and overdispersion.

Table 11. Summary of Results from Lagged Models for Daily Mortality

Age	Mortality Measure	Longest Positive Cumulative Lag (p<.05)							
	Kuala Lumpur PM <sub>10</sub>								
All	Total	1 day							
65-74	Total	5 days							
65-74	Non-Traumatic	9 days							
65-74	Other	4 days							
Kuala Lumpur Visibility									
All	Total	1 day							
All	Non-Traumatic	1 day							
All	Other	1 day							
< 1	Total	1 day							
< 1	Non-Traumatic	1 day							
65-74	Total	5 days							
65-74	Non-Traumatic	5 days							
65-74	Cardiovascular	6 days							
	Kuching Visib	ility							
All	Respiratory	2 days							
75+	Total	1 day							
75+	Non-Traumatic	1 day							
75+	Cardiovascular	1 day							

Note: Models control for weather conditions (temperature and humidity), a time trend (capturing changes in population and mortality), and overdispersion.

Table 12. Cumulative Lagged Effects of  $PM_{10}$  for Daily Mortality in Kuala Lumpur Due to Non-Traumatic Causes, Ages 65-74, by Length of Lag

Lag	Cumulative Effect
28	1.436
27	1.412
26	1.080
25	1.195
24	1.257
23	1.545
22	1.547
21	1.619
20	1.752*
19	1.795*
18	1.948*
17	1.793*
16	1.636
15	1.702*
14	1.508
13	1.530
12	1.534
11	1.547
10	1.714*
9	1.790**
8	1.831**
7	1.831**
6	1.848**
5	1.978**
4	2.186***
3	1.941**
2	1.738**
1	1.716***

Notes: \* p<.10; \*\* p<.05; \*\*\*p<.01.

Models control for weather conditions (temperature and humidity), a time trend (capturing changes in population and mortality), and overdispersion.

Figure 1. PM<sub>10</sub> Concentration for Kuala Lumpur, Malaysia, 1996-97

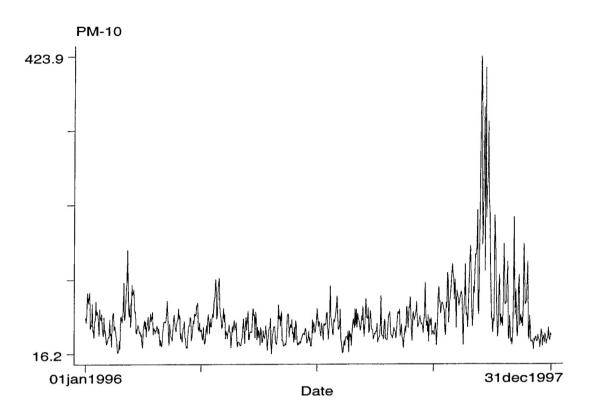


Figure 2. Visibility for Kuala Lumpur, Malaysia, 1994-97

